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EELGRASS WASTING DISEASE: CAUSE AND RECURRENCE OF A MARINE EPIDEMIC

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Abstract

Eelgrass populations are currently infected with a disease that produces symptoms and epidemiology reminiscent of the famous eelgrass wasting disease of the 1930s. This disease virtually eliminated eelgrass (*Zostera marina* L.) in the North Atlantic for three decades. For 50 years scientists have speculated about the cause of the 1930s eelgrass decline. We have now proven that the causal organism of the present epidemic is a pathogenic strain of *Labyrinthula,* which was suspected, but never conclusively shown to cause the 1930s wasting disease. We have isolated the infectious form of *Labyrinthula* from eelgrass from Maine to North Carolina on the Atlantic coast, and from Puget Sound on the Pacific coast; disease-related dieoffs of eelgrass beds are confirmed in Maine, New Hampshire, and Massachusetts.

Discussion

A recurrence of the wasting disease, which almost eliminated eelgrass (*Zostera marina* L.) in the 1930s, was discovered on the border of New Hampshire and Maine in the early 1980s (Short *et al.,* 1986). Since then, eelgrass populations exhibiting symptoms and epidemiology comparable to the 1930s epidemic have been found from Nova Scotia to North Carolina. The eelgrass wasting disease of the 1930s constituted a marine epidemic which disrupted highly productive coastal ecosystems and fisheries. The disease had run its course by the 1940s; healthy eelgrass populations generally were reestablished by the 1960s. Over the past 50 years, scientists have proposed pathogenic organisms, temperature changes, reduced light, and combined environmental factors as causes of the 1930s disappearance of eelgrass. In this report, we present proof that the causal organism of the current epidemic is a pathogenic strain of *Labyrinthula* and describe our findings concerning the range of the disease.

The wasting disease that devastated eelgrass populations throughout the North Atlantic between 1930 and 1933 dramatically disrupted coastal and nearshore ecosystems. The most obvious impact was the reduction or loss of migratory waterfowl populations (Addy and Aylward, 1944). Equally important, though not immediately apparent, was the impact on commercial fisheries. The loss of the scallop fishery in the mid-Atlantic coast of the United States is best documented (Thayer *et al.,* 1984). The 1930s eelgrass loss altered coastal habitats and changed for decades the characteristics of nearshore soft sediment environments (Rasmussen, 1973, 1977). In fact, some locations were permanently altered, and eelgrass never returned.

The cause of the 1930s wasting disease was never conclusively determined (Johnson and Sparrow, 1961; Den Hartog, 1987). However, two main alternative theories

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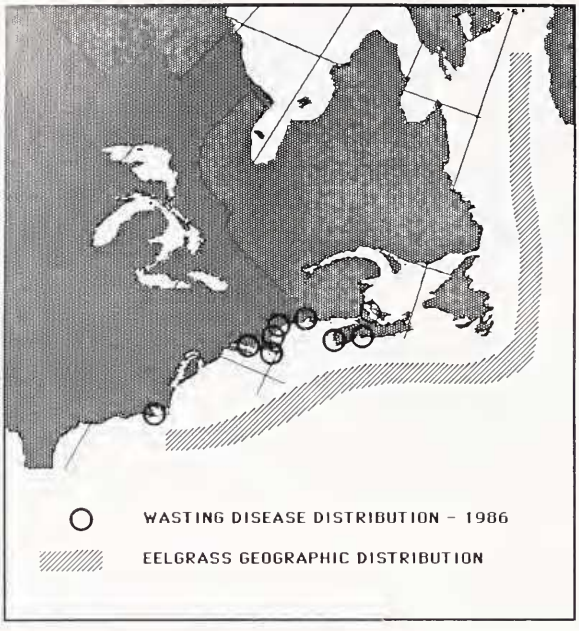

FIGURE 1. Geographic distribution of eelgrass and of the current wasting disease on the east coast of North America.

were promoted: first, that a microorganism was infecting and killing plants (Peterson, 1934; Renn, 1935), and second, that environmental stress from abnormally warm temperatures increased the susceptibility of the plants to ever-present microorganisms (Rasmussen, 1977). The microorganism most commonly implicated was a slime-mold-like protist, *Labyrinthula* (Renn, 1934; Cottam and Addy, 1947), identified as *L. macrocystis* Cienk. (Young, 1943). In the decades since the first reports of the wasting disease there has been significant organism-specific research on *Labyrinthula* (Pokorny, 1967; Olive, 1975; Porter, 1988). The early work during the disease period was not conclusive because the necessary methods for axenic culture of *Labyrinthula* had not been developed (Renn, 1936; Johnson and Sparrow, 1961). Although axenic cultures of *Labyrinthula* (Watson and Ordal, 1957) were developed in the 1950s, tests of Koch's postulates were never attempted.

The current eelgrass wasting disease, first reported in the Great Bay Estuary, New Hampshire (Short *et al.*, 1986), occurs in two stages: (1) the initial infection of eelgrass leaves; and (2) the subsequent mass mortality of eelgrass. The infection is characterized by dark necrotic lesions on both young and old eelgrass leaves. The infection has now spread throughout the Great Bay Estuary, but the complete dieoff of eelgrass beds is restricted to local areas. Like the disease of the 1930s, the current epidemic is limited in the estuary by salinity; eelgrass growing in low salinity waters is less susceptible to infection. The decline is not universal; many areas showing infection symptoms as yet demonstrate no mass mortality.

The infection of eelgrass with the characteristic symptoms of the wasting disease is now widespread along the Atlantic coast of North America. Eelgrass collected in the summers of 1986 and 1987 from numerous sites between Nova Scotia, Canada,

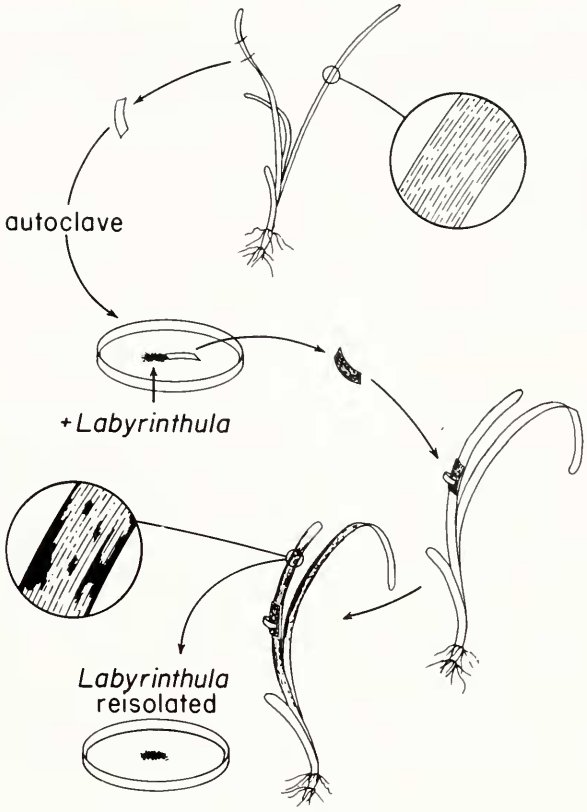

FIGURE 2. Procedure for laboratory infection of eelgrass, *Zostera marina* L., by *Labyrinthula*. Sterilized 1 cm pieces of uninfected, green eelgrass leaves were invaded with an axenic culture of *Labyrinthula* and then attached to a leaf of a healthy, green eelgrass shoot growing in an individual incubation flask. When a pathogenic strain was thus tested, the necrotic, blackened patches symptomatic of the wasting disease appeared on the eelgrass leaves within 14 h on some and within 24 h on all the plants. *Labyrinthula* was reisolated from the diseased leaves, thus satisfying Koch's postulates.

and Connecticut, USA, showed the infection (Fig. 1). Additionally, in 1986 infected eelgrass plants were found in Roscoff, France, and in Friday Harbor, Washington, and Beaufort, North Carolina, USA. The simultaneous occurrence of the wasting disease on both sides of the Atlantic is reminiscent of the reports of the 1930s disease (Fisher-Piette *et al.,* 1932; Huntsman, 1932; Cotton, 1933; Lewis and Taylor, 1933; Peterson, 1933; Taylor, 1933). The appearance of the disease on the Pacific coast was reported in the late 1930s (Young, 1938), with significant eelgrass decline evident in 1941 (Moffit and Cottam, 1941).

Our recent research has concentrated on determining the cause of the current eelgrass disease. Following Koch's postulates, we have successfully identified the causal agent to be a pathogenic strain of *Labyrinthula* (Fig. 2). We have regularly isolated this strain of *Labyrinthula* from diseased eelgrass leaves from Great Bay, New Hampshire and also from Beaufort, North Carolina and Friday Harbor, Washington. The pathogenic strain has never been isolated from healthy, green eelgrass

TABLE I

Labyrinthula infection experiments on eelgrass (Zostera marina) *in laboratory culture*

Labyrinthula source: Substrate, location	Number of replicates	Percent infection
Diseased eelgrass, Great Bay, NH	33	100
Diseased eelgrass, Puget Sound, WA	8	100
Diseased eelgrass, Beaufort, NC	4	100
Healthy eelgrass, Beaufort, NC	4	0
Spartina, Sapelo Is., GA	6	0
Codium drift, Weekapaug, RI	9	0
Mangrove leaf, Longboat Key, FL	7	0

leaves. Both pathogenic and non-pathogenic strains of *Labyrinthula* were isolated and then grown in pure culture using previously described procedures (Porter, 1988). The eelgrass infection experiments were performed in laboratories at both the University of Georgia and the University of New Hampshire (Table I). Every shoot exposed to the pathogenic strain of *Labyrinthula* (45 shoots of a total of 45) exhibited the disease symptoms. None of the nine control shoots, which were treated identically, but without *Labyrinthula* in the sterilized inoculum leaf piece, showed disease symptoms; all remained healthy. During the first week, the infection spread quickly, with the dark patches increasing to 3–4 cm lesions along the inoculated leaves. After two weeks, inoculated leaves were completely black or brown and, on most shoots, the infection had spread to other leaves. After three weeks, several of the infected shoots were completely brown and dead, while on other shoots the spread of the infection stopped; the growth of all the infected plants was greatly reduced relative to the control plants. *Labyrinthula* was reisolated from the infected leaves, thus satisfying Koch's postulates.

For the present study, four other strains of *Labyrinthula* were isolated from green eelgrass leaves and various other marine plants. When these strains were tested for pathogenicity, as described above, none of the 26 inoculated plants produced any signs of the disease symptoms (Table I).

It is significant that we found both pathogenic and non-pathogenic strains of *Labyrinthula* widely distributed in estuarine environments. It is possible that these represent different species, but the present taxonomic understanding of the species *Labyrinthula* is poor at best (Olive, 1975; Porter, 1988). A critical monograph of the genus is clearly needed.

Despite the widespread infection of eelgrass with the wasting disease, there is as yet relatively little documented evidence of disease-related declines. As mentioned above, the carefully monitored decline in the Great Bay Estuary has expanded (Short *et al.*, 1986). Since 1984, entire eelgrass beds have died and large portions of other beds have disappeared. A nearly complete decline of eelgrass at Cape Ann, Massachusetts in 1984 (Dexter, 1985), has been linked to disease through subsequent sampling of a few remnant eelgrass plants which proved to be infected.

Other declines in eelgrass have been reported. Loss of eelgrass from estuaries in both North America and Europe has been shown to result from pollution of coastal regions (Jones and Tippie, 1983; Kemp *et al.*, 1983; Neinhuis, 1983; Orth and Moore, 1983). Elsewhere in the world, other seagrasses have also experienced pollution-related declines (Cambridge and McComb, 1984). Loss of eelgrass was reported from several other areas along the east coast of the U. S. in 1986, including Buzzards Bay

and Cape Cod, Massachusetts, where the cause was pollution and Great South Bay, New York, where the cause was shading by a plankton bloom. Although the wasting disease has also been discovered in some of these locations, there is no evidence that it has contributed to any of these declines. However, we believe that the combined effects of the wasting disease and pollution could devastate eelgrass populations.

Whether the current eelgrass wasting disease will produce a catastrophic eelgrass decline is unknown. Although the current wasting disease has not yet caused a widespread decline of eelgrass populations, the disease poses a major threat to coastal fisheries, waterfowl populations, numerous marine habitats, and the health of estuarine environments. What has produced the recurrence of this widespread epidemic, what circumstances might bring this disease to the stage that devastates eelgrass populations, and what role environmental factors may play in this transition remain matters of conjecture and further investigation.

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